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GJS:CJL

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BIOLOGICAL AND CHEMICAL RESEARCH INSTITUTE, RYDALMERE

Dr. J. Lederberg
Department of Genetics
University of Wisconsin
Madison. Wisconsin. U.S.A.

21st August, 1973

Shanahan
file 3/74

Dear Dr. Lederberg,

Please be tolerant with this request for a comment. The enquiry concerns the origin of resistance in insects, in which it is now reasonably well established that single genes are almost invariably involved - more than one gene may give resistance to some chemical but each allele is an oligogene.

Your conjoint article with Cavalli-Sforza on pre-adaptive resistance to streptomycin in E. coli has always been a guide, despite the difference in the type of organism involved, in regard to the origin of resistance in insects, ticks and mites. Needless to add the great differences involved - haploid versus diploid - remove strict comparison but this may not be fully so if attention is directed to primordial sex cells in say, flies, especially to sperm, which can occur in quantity and provide a relatively large population on which a "systemic" insecticide such as dieldrin can exert pressure due to its delayed action in larvae - brief immersion of young larvae in dieldrin emulsion, e.g. can give full emergence of flies which die shortly after. With this introduction, a couple of questions: it is wondered why the rate of 100 ug/ml of streptomycin was used as the diagnostic dose to diagnose resistance; next, whilst it is appreciated that stremptomycin resistance is a single step condition, is it definite (excuse the post-adaptive implication) that exposure of E. coli to non-toxic levels, say 10 ug/ml, does not prepare cells for an expression of streptomycin resistance?

SHANAHAN, G.

In laboratory selection work with flies, etc., commencing with known susceptibles, resistance has been often obtained. This seems odd and it has been raised with Professor Crow. Since single genes are involved in resistance a closed population of flies or mosquitoes is compared for the purpose of this exercise with a closed flock of polled sheep, which remain hornless generation after generation.

I might add that I have been working on resistance in insects for some 20 years - its origin remains a mystery, not so much in regard to its appearance in the field, but in closed colonies. It is quite easy to see at normal mutation rates that pre-existent R gene(s), as you established, explains resistance to bactericides but the origin of resistance in small colonies of insects pressurised with an insecticide seems to be an extreme expectation. Incidentally, whilst the normal mutation rate for R allele to an insecticide is largely unknown, it must not be high for rr colonies seem to remain homozygous.

Enclosed please find a reprint on the genetics of dieldrin resistance in Lucilia.

If Professor Crow is still at Madison please give him my regards.

I would be very grateful for your comments.

Yours sincerely,

G.J. SHANAHAN.